FADING ELK SYNDROME – AETIOLOGY

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Abstract
A survey of Wapiti farmers in 2008 revealed high levels of Fading Elk Syndrome. A study of affected animals from 9 farms confirmed abomasal parasitism in all cases. A scoring system was developed based on a range of histological changes occurring from parasite damage in the abomasa. Issues with anthelmintic application method and or drench resistance are suggested and do explain the increased incidence in recent years. Effective anthelmintics must be used and adult Wapiti and Elk need targeted at times of stress.

Keywords
Wapiti, elk, abomasal parasitism, Ostertagia, moxidectin, efficacy

Introduction
Fading Elk Syndrome has plagued the farming of Wapiti and Elk in New Zealand for as long as deer have been farmed here – recorded from the mid 1980's through to today. (Bringans 1987, Pearse 1988, Mackintosh and Orr 1990, Conway 1990, Scott 1992, Waldrop and Mackintosh 1992, Dougherty 2008) The syndrome has recently been recorded in North America (Woodbury and Parry 2009)

Typically these deer are older than yearlings, loose condition and become emaciated, may or may not scour, have a decreased appetite, often appear vacant, dopey and starry-eyed. They tend to have a typical gait, running with a lower head carriage, almost uncoordinated limbs – "a plonking" gait. They normally seem refractory to all or any treatments but survive in this state for some months prior to dying.

The cause of Fading Elk Syndrome has been variously linked with nutrition, copper other trace elements and vitamins, fungal toxins, infectious agents, pasture, genetics and parasitism. With this plethora of causes postulated over the years, it is not surprising that a "shotgun" approach to treatment has been the "norm". The role of abomasal parasitism particularly Ostertagia gained significant traction following the work of Waldrop and Mackintosh 1992.

Background
Anecdotal comment from farmer members of the Elk and Wapiti Society of New Zealand (EWSNZ) in 2008 indicated it had been a particularly bad year for the incidence of Fading Elk Syndrome (FES). In response the council of EWSNZ decided to survey members to get a better idea of the scope of the problem and also to hopefully get some directive as to where they could consider looking for a resolution/s to the issue. The author being a founding member of the society and wapiti farmer was charged with the task.

We achieved a 55.5% response from farmer members and this represented some 16,000 farmed Wapiti on 25 farms from throughout New Zealand.

FES occurred on 19 of the farms in 2008 with an average incidence of 6.2% (range 0.1% to 24%). A total of 392 animals were affected with 241 of these subsequently dying. Death rates/survival rates varied from 0% to 100% between farms.

Across all farms deer with higher proportion of elk genes were more affected. This is consistent with previous reports (Pearse 1988, Mackintosh and Orr 1990, Conway 1990, Scott 1992, Dougherty 2008)
Onset of FES was observed in the Autumn in 60% of cases with the remainder in Winter. In difference to other years the summer of 2007/08 across almost all farms had been dry to the extent of affecting feed quality and pasture cover.
No correlation in incidence was found with integrated farming systems, standard of handling facility, regrassing policy in the previous decade or vaccine use.
Autopsies had been carried out on only 4 of the 241 deaths from FES (perhaps a sad reflection on the veterinary profession). 1 was too autolysed and the other three all indicated evidence of abomasal parasitism.
The survey question on treatment and response was difficult to interpret. Based on farms with no incidence of FES or where reasonable success had been achieved with treatment we cautiously recommended use of the combination drench – Cydectin Inj and oral Scanda

Material and methods
Armed with the survey information the EWSNZ developed a plan to investigate FES further. The aim of the study was to determine if Ostertagia was the cause of FES and at the same time to see if drench resistance was present and/or contributing.
Eighteen deer spread over 9 farms in the Otago/Southland region were identified as chronically affected with FES and the owners were prepared to sacrifice these deer.
All deer were euthanased in August 2009 with onset of symptoms recorded in the Autumn of 2009 except for 4 deer where the onset had been noted in Autumn 2008.

Sixteen of the18 animals were weighed and drenched at least 7 days prior to euthanasia

They were all euthanased and samples collected

- Brain/brain stem fixed and fresh section spinal cord for routine TSE surveillance targeting CWD
- Whole abomasa fresh and chilled for pH, total worm counts, abomasal digest counts and histology
- Liver and jejunal sections fixed for histology.

At the time of autopsy gross assessment was made regarding the lungworm and Johnes status of the deer.
All samples were couriered overnight and processed appropriately at Gribbles Invermay lab well inside 24hrs of euthanasia.
Dr John Gill (Gribbles Invermay pathologist) from the histology developed a comprehensive scoring system based on an estimate of the severity of parasite damage to abomasal folds. A scale of 1-10 (1 being not affected and 10 severely affected)
Factors included average depth of mucosa (max and min in microns), number of dilated crypts/fold, numbers of nematode larvae in mucosa/fold, numbers of adult nematodes in lumen/fold, Inflammatory cells/ 10 hpf (lymphocytes, eosinophils, neutrophils, plasma cells), Lymphoid hyperplasia, extent of parietal cell depletion and the extent of crypt mucous metaplasia/hyperplasia of crypt epithelium.

Results
All deer met the criteria on the TSE Surveillance Submission Form of "Progressive non-responsive case of ill-thrift" with only some partially showing "Progressive non-responsive nervous disease" All samples submitted for CWD had negative results.

At autopsy 2 deer showed gross lesions consistent with Johnes disease both of which were confirmed by histology.
All deer except one on Farm B* (see Table 1) had elevated abomasal pH and high abomasal histology scores consistent with severe parasite damage.

Table1: Histology Scores and pH from deer with Fading Elk Syndrome

Farm	Age Years	Sex	Abomasum pH	Abomasum Histol Score	Johnes gross	Johnes histol
A	9	F	6.1	7	-	-
A	6	F	6.1	7	-	-
B*	2	M	3.3*	3*	+ve	+ve
C	8	F	6.2	8	-	-
C	8	F	6.6	6	-	-
D	2	F	5.7	8	-	-
D	7	F	6.1	6	+ve	+ve
E	2	F	6.5	5	-	-
E	10	F	6.3	8	-	-
F	6	F	6.4	8	-	-
F	2	F	6.8	9	-	-
G	3	M	6.6	7	-	-
G	3	M	6.8	ND	-	-
H	7	F	ND	9	-	-
H	9	M	ND	7	-	-
J	2	F	ND	6	-	-
J	1	F	ND	5	-	-
J	1	F	5.5	5	-	-

All but two of the affected deer were treated with moxidectin as Pour On or Injection within 30dys of euthanasia. With the exception of these 2 deer no lungworm were observed at the time of autopsy. Significant numbers of Ostertagia adult and larva were recovered from these animals particularly the Pour On treated animals (see Table 2)

Table 2: Post treatment parasite burdens from deer with Fading Elk Syndrome

Farm	Score	Drench date	Drench type	Worm count Ostert	Worm count Tric	Digest Ostert	Digest Tric	Lungworm gross
F	8	>4mths	Cyd PO	13800	16000	800	300	moderate
F	9	<30dys	Cyd PO	400	-	-	-	-
G	7	4wks	Cyd PO	18500	2500	2000	100	-
G	ND	4wks	Cyd PO	6600	2000	1000	-	-
H	9	5mths	Cyd PO	8700	16700	-	1000	mild
H	7	9dys	Cyd PO	1800	4100	-	2700	-
J	7	13dys	Cyd PO	6400	500	-	2700	-
J	5	13dys	Cyd PO	2300	500	ND	ND	-
J	5	8dys	Cyd inj	-	-	100	-	-
A	7	7dys	Cyd inj	-	-	-	-	-
A	7	7dys	Cyd inj	2300	-	-	-	-
B	3	8dys	Cyd inj	100	-	200	-	-
C	8	8dys	Cyd inj	-	-	-	-	-
C	6	8dys	Cyd inj	-	-	300	-	-
D	8	7dys	Cyd inj	1000	-	-	-	-
D	6	7dys	Cyd inj	-	-	-	-	-
E	5	12dys	Cyd inj	-	-	-	-	-
E	8	12dys	Cyd inj	-	-	-	-	-

Jejunal histology showed some evidence of parasitic migration in two animals and another two with pathology consistent with Johnes, otherwise no significant findings. Liver sections revealed some mild changes in5 animals by way of dilation of central veins and thickening of the wall.

Discussion

The elevation in abomasal pH as a result of the depletion of parietal cells following Ostertagia infection has been documented (Connan 1991, Waldrup and Mackintosh 1992).
All 18 deer (with one exception) had elevated abomasal pH and high histological scores indicative of severe parasitic damage.
The one exception on Farm B had minimal parasite damage but was confirmed with Johnes disease. This provided us with a reference pH close to normal. Normal being <3 (Mackintosh pers commun) and served us with a differential diagnosis reminder for animals with these clinical symptoms.

Moxidectin Pour On had been shown to have >99.9% efficacy against mature and immature Ostertagia-type nematodes (Waldrup et al., 1998)
There were no “control” animals in the sample group and so we do not know the pre-treatment gastro-intestinal parasite status of these deer. Technical comment from the Moxidectin manufacturer (Pfizer), suggest that due to the mechanism of action of the

drug on the parasite we should have euthanased at least 12days post treatment. Hence our numbers may be overstated in some instances. Notwithstanding these factors the expectation would be of a close to zero worm count from the moxidectin treated deer. A far different but fairly clear picture emerged – the level of Ostertagia and Trichostrongyles (adult and larva) in the abomasa of Moxidectin Injection treated deer was much less than that of Moxidectin Pour On treated deer. Do in fact we have drench resistance here? At very least there is an apparent discrepancy in efficacy based on application method

The wisdom of hindsight highlights the role that abomasal parasitism plays in Fading Elk Syndrome. From the perspective of a practice deer veterinarian, in the 1980's we had BZ's but they were not proving satisfactory in prevention or treatment of lungworm which was considered "the" parasite issue of farmed deer. The incidence of FES seemed to be increasing. Along came the Ivomec (first of the ML's) and in practice we found doubling the standard Pour On dose was helpful with FES. The real break through came with the advent of Moxidectin Pour On. By the mid 1990's we looked to have FES beat. Then over the next approx 15yrs (Moxidectin became almost exclusively used on most deer farms (Castillo-Alcala et al 2005) we saw a gradual increase in the incidence of FES coming to a head in 2008 (as reflected in the survey). The level of moxidectin drench resistance developed over this time has escalated (Waldrup et al., 1998, Hoskin et al., 2005, Pomroy 2006, Lawrence 2011 these proceedings).

Considerable time, effort and expense has occurred over the last two decades with investigations into the role in FES of Copper, Vitamins other trace elements and minerals etc All efforts in future need simply be directed at an effective anthelmintic for mature and immature forms of Ostertagia.

Anecdotal comment from Wapiti farmers would indicate that combination drenching (Moxidectin Inj plus an Oral BZ/levamisole combination) has all but eliminated FES in the last 2 yrs on their farms.

Follow up has occurred on small numbers of FES survivors and it may be there are issues with the ability of Wapiti and Elk type deer to repair and regenerate the abomasal lining.

Conclusion

We now have compelling evidence that Fading Elk Syndrome is caused by abomasal parasitism.
The epidemic type incidence of FES in 2008 now has a plausible explanation. Although unrecognized at the time there would have been resistance to moxidectin present. An ineffective anthelmintic plus a season providing a much greater Ostertagia challenge than normal was responsible.
Elk and Wapiti type deer appear not to develop a strong immunity to gastrointestinal parasites with age. Consequently are susceptible at times of stress (the roar for stags and lactation for hinds) which also coincides with peek pasture parasite challenge in the Autumn (Connan 1996)

Strategic use of an anthelmintic with good efficacy against mature and immature Ostertagia-type nematodes will prevent Fading Elk Syndrome

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